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Modern Concepts of Cardiovascular Disease

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DR. JAMES G. CARR, Chicago, *Editor*

DR. GILBERT H. MARQUARDT, Chicago, *Associate Editor*

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ANGINA PECTORIS, CORONARY FAILURE AND MYOCARDIAL INFARCTION: THE CLINICAL IMPORTANCE OF INTERCORONARY COLLATERAL CIRCULATION

The interpretation of various conditions in which cardiac pain is the primary symptom and the diagnostic terms denoting these syndromes are subject to considerable diversity of opinion. Some clinicians have urged abandonment of the clinical diagnosis of angina pectoris with substitution of a physiologic concept, "coronary insufficiency." Others characterize all episodes of prolonged cardiac pain by such terms as "coronary thrombosis," "coronary occlusion" or "myocardial infarction." Because of the frequent disparities between the clinical prediction and the pathologic observations, a detailed clinical and pathologic study was made in a consecutive series of necropsies. The clinical diagnosis of "coronary thrombosis" is often made but no such thrombosis may be found; in other instances a fresh cardiac infarct is disclosed but no fresh thrombosis of any arteries supplying the affected area may be evident. Conversely, fresh coronary thrombosis or complete occlusions of main coronary arteries may be found postmortem but without any corresponding antecedent clinical phenomena.

In the pathologic examination of the heart, the injection plus dissection method devised by one of us (M.J.S.) was used. According to this technique the right and left coronary arteries are injected simultaneously with differently colored radio-opaque masses under a pressure of from 150 to 200 mm. of mercury. The heart is then unrolled so that all of the coronary arteries lie on one plane and a roentgenogram is made. A complete dissection of the arteries is then carried out. Multiple representative sections of the myocardium are studied histologically.

The 355 consecutive necropsied cases fall naturally into the following categories: (1) patients with no clinical evidence of cardiovascular disease; (2) patients with angina pectoris; (3) patients with coronary failure, i.e., pain more prolonged than that denoted by the term angina pectoris, but not associated with clinical evidence of myocardial necrosis; and (4) patients with acute myocardial infarction, i.e., prolonged pain plus evidences of myocardial damage, namely, fever, leukocytosis, elevated corrected sedimentation rate and progressive electrocardiographic changes.

Observations in Patients with No Clinical Evidence of Cardiovascular Disease. Watery solutions injected into one coronary artery are always found in the other large coronary arteries, demonstrating that even in the normal heart fine communications exist between the main coronary arteries. When, however, normal hearts were injected with the colored lead agar mass used in these studies, passage of the mass from one coronary artery to the other side was unusual. Since the lead agar suspension penetrates regularly only as far as arterioles 40 micra in diameter, interarterial collateral pathways of 40 micra or more may be regarded as generally absent in normal hearts. If narrowing of a coronary artery occurs, these larger collateral pathways of 40 or more micra may develop and supply the area of the myocardium which would otherwise suffer from diminished blood flow. The collateral pathways which hitherto have attracted particular attention communicate between arteries and the cavities of the heart or between capillaries, veins and the cavities of the heart. The collateral pathways demonstrated in abnormal hearts by the lead agar mass communicate between the major coronary arteries themselves and appear to be of considerable importance in obviating the effects of arterial narrowings and occlusions.

It has been of particular interest to note the absence of collateral pathways in many normal hearts with little or no coronary arteriosclerosis or other abnormalities in patients over 70 years of age. This indicates that the development of an anastomotic circulation is not a necessary concomitant of the aging process, but is rather to be related to the development of narrowing or occlusion of an artery. This conclusion is somewhat contrary to the experience of others. In some hearts, collateral pathways were observed in the presence of myocardial hypertrophy or valvular lesions in the absence of coronary arterial occlusions.

Complete occlusion or considerable narrowing of one or more coronary arteries can exist without giving rise to any clinical symptoms or signs and may be compatible with comfort and longevity. Thirty-eight cases with old occlusions of the main coronary arteries or primary branches were found without related clinical signs or symptoms; twenty-five showed complete occlusion of at least one main coronary artery; thirteen showed complete occlusion of one or more primary branches. In accord with the absence of angina pectoris and congestive failure in these cases was the infrequent occurrence of advanced myocardial damage.

This apparent inconsistency between the presence of long-standing obstructive arterial lesions and the absence of significant pathologic or clinical evidence of myocardial damage was dispelled by the demonstration of a collateral circulation in relation to the obstructions in each of these hearts. In some hearts the collateral pathways communicated between portions of the same artery proximal and distal to the occlusion; in other instances the artery distal to the occlusion derived its supply from distant vessels. The collateral circulation may continue to safeguard the heart from serious myocardial damage, but the

sudden development of an occlusion may result in acute myocardial infarction if time is insufficient for the development of further anastomotic vessels.

Observations in Patients with Angina Pectoris. Every patient suffering primarily from angina pectoris without evidence of valvular disease or of arterial hypertension has shown at postmortem examination old complete occlusion of at least one major coronary artery; in the majority of instances at least two of the three main coronary arteries had been occluded before the terminal illness.

While there is a general relationship between the incidence of coronary occlusions and the occurrence of angina pectoris, other modifying factors such as the exact site of the occlusion, the importance of the vessel involved, the adequacy of the collateral circulation, the rate at which such occlusions or narrowings develop, the influence of emotion and of vasomotor reflexes are also of great importance. In patients, previously well, who suddenly suffer attacks of mild or severe angina pectoris on but little exertion, the occurrence of rapidly progressive narrowing or of sudden complete occlusion of a coronary artery must be considered. A similar interpretation must be entertained in patients with long-standing angina pectoris if attacks are suddenly more readily precipitated. Under such circumstances bed rest is strongly advisable to decrease the demands on the heart while there is developing a more adequate collateral circulation.

Observations in Patients with Coronary Failure. In certain cases, cardiac pain more prolonged than that consistent with angina pectoris has been noted, but the clinical evidences of myocardial necrosis, such as fever, leukocytosis, increased sedimentation rate and progressive electrocardiographic changes over a period of days, were not observed. The hearts of such patients did not show myocardial infarction on the occasions when they were examined postmortem. In accord with the concept that cardiac pain is due to relative myocardial ischemia, such attacks usually were coincident with increased demands on the heart, such as paroxysmal heart action, emotion or exertion, whereas in other instances decreased coronary blood flow following fall of blood pressure in shock was apparently responsible. These episodes of coronary failure differ from angina pectoris in the longer duration of the attack and in some instances in the altered characteristics of the pain. On the other hand, the signs of myocardial necrosis are absent. The clinical diagnosis of either angina pectoris or acute myocardial infarction would be erroneous.

Observations in Patients with Acute Myocardial Infarction. The clinical diagnosis of acute myocardial infarction is based on the existence of prolonged cardiac pain plus clinical evidences of myocardial necrosis. These consist of fever, leukocytosis, increased sedimentation rate and electrocardiographic changes progressing over a period of days. Acute myocardial infarction may occur without a concomitant fresh thrombosis; and, contrariwise, thrombosis of a major coronary artery may occur without causing acute myocardial infarction.

The results of these studies indicate that the disparity between clinical diagnosis and the pathologic condition in the heart is largely due to the fact that the terms coronary thrombosis and coronary occlusion actually refer to a pathologic event which may give rise to various clinical syndromes or, indeed, to no clinical symptoms or signs. The present studies indicate that there is no characteristic syndrome necessarily associated with coronary arterial occlusion *per se*. In the past the terms "coronary thrombosis" and "coronary occlusion" have been used by many clinicians as being synonymous with acute myocardial infarction. Our observations as well as those by others demonstrate, however, that acute myocardial infarction is not necessarily related to the formation of thrombi or occlusions; either event may occur in the absence of the other. In our series, twelve of forty-five hearts with acute myocardial infarction showed neither acute coronary thrombosis nor other acute occlusion. The use of the term "acute coronary occlusion" to designate the existence of acute myocardial infarction would have been inaccurate in 27 per cent of the cases of infarction; the term "acute coronary thrombosis" would have been inaccurate in 47 per cent. Conversely, twenty-six hearts showed old complete occlusions without any myocardial infarction. Eleven additional hearts had old occlusions and fresh myocardial infarction but no fresh occlusions; here again the terms "acute coronary thrombosis" or "acute coronary occlusion" would have been incorrect.

The clinical use of the three terms *angina pectoris, coronary failure* and *acute myocardial infarction* to differentiate the various syndromes in which attacks of cardiac pain are witnessed is a modification of terminology hitherto employed and conforms more accurately to the observed conditions found. Our data are in accord with the current belief that cardiac pain is a manifestation of myocardial ischemia. In all three of the aforementioned syndromes, i.e., angina pectoris, coronary failure and acute myocardial infarction, the underlying mechanism seems to be a relative disproportion between the demands of the myocardium for blood and the supply of blood through the coronary arteries. The changes in the myocardium resulting from this disproportion depend solely on the extent and duration of the relative ischemia, not on the manner by which it is produced. If the duration of the ischemia, as in angina pectoris, is brief, no permanent myocardial damage may occur or only microscopic foci of necrosis and subsequent fibrosis may result. Even with more prolonged attacks of ischemia and pain, as in coronary failure, if the demands on the myocardium are quickly reduced by rest and sedatives or the control of abnormal cardiac rhythm, no more extensive permanent structural damage may result. Clinical evidence as well as experimental observations on the effects of temporary coronary occlusion conform to these considerations.

Herrman L. Blumgart, M.D.
Monroe J. Schlesinger, M.D.
Paul M. Zoll, M.D.
Boston, Mass.

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